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PMS IN THE WORKPLACE: MYTH OR METHOD?

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Hardie (1997) purportedly examined the effects of menstrual cycle status and perceived (i.e., self-diagnosed) premenstrual syndrome on work performance. Although research to fill the numerous gaps in our understanding of PMS is encouraged, methodologic imprecision compromises Hardie's findings.

Hardie (1997), in an article titled "PMS in the Workplace: Dispelling the Myth of Cyclic Dysfunction," claimed to provide evidence that menstrual cycle status has no effect on female employees' work performance. In doing so, she opened and closed her presentation by referencing our earlier work, published in this journal, in which we (Phillips & Bedeian, 1989) offered that there was yet clear-cut scientific evidence that premenstrual syndrome (PMS) adversely affects workplace behavior. She further stated that we nonetheless held that because "most people believe the opposite, employers should acknowledge PMS as a workplace problem" (p. 97). The purpose of this brief commentary is to (a) correct Hardie's misinterpretation of our work and (b) proffer that, despite Hardie's findings, the question of whether PMS influences job performance remains unanswered. Our aim is not to defend one position or another. Rather, it is to consider methodological inadequacies in Hardie's study design that render her find-

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ings problematic. It is our belief that dialogues of this nature are useful for highlighting methodological concerns that have characterized research in this area and, thus, for advancing our knowledge base.

Misinterpretation

Hardie misinterprets/mis-represents our work on two counts. First, contrary to Hardie's contention, we did not state that "most people" believe PMS affects employee behavior. We made no such relative assessment. Second, we did not then conclude that, for this reason, "employers should acknowledge PMS as a workplace problem." We did, however, hold that because "women and men alike believe there are tangible associated effects . . . it seems logical to approach PMS-related beliefs as attributions" (p. 171). In specifying an agenda for PMS research we did go on to call for future investigations into whether such attributions (a) differentially affect male and female evaluations of female employee performance and (b) apply to women as a group or only to specific women (i.e., those exhibiting PMS symptoms). Thus, counter to Hardie's claim, we did not suggest that employers acknowledge PMS as a workplace problem, but did conclude that the belief or attribution associated with the perception that PMS effects job performance is a problem that should be addressed. Indeed, Hardie's data, collected using perceptual-based (i.e., self-diagnosed) measures of PMS and menstrual cycle status, support this conclusion. The perceived PMS (PPMS) group in her study "consistently" reported greater work impairment than a No PPMS comparison group, despite there being no between-group differences in their work performance. As Hardie states, "for female employees, the perception of having a cyclic disorder was itself problematic" (p. 101). The use of our work to formulate a straw man, for whatever purpose, is rejected.

METHODOLOGICAL CONCERNS

It is also Hardie's claim that whereas the bulk of early PMS research was conceptually and methodologically flawed, thereby contributing to the myth of widespread cyclic dysfunction, her findings dispel this myth. This claim aside, several methodological aspects of Hardie's study are of concern. Our intent is not to criticize Hardie per se, but to consider aspects of her study in light of difficulties that may be particularly salient in future studies into the effects of PMS on workplace behavior.

Sample Frame

The nature of a study's sample frame should clearly define the focal groups to be investigated. In this respect, Hardie addresses neither the representativeness nor relevance of the 438 university employees (95 men, 343 women) she sur-

veyed to gather the data on which her results are based. To be sure, the adequacy of entry criteria in screening a target population is confounded by a multiplicity of factors common to all PMS research. First among these factors is that both the very definition and diagnosis of PMS are widely contested (Halbreich & Endicott, 1985). The aetiology of PMS is likewise debated. Broadly considered, PMS may be prototypically defined as "the cyclic recurrence, in the luteal phase of the menstrual cycle [i.e., the week before the onset of menses] of a combination of distressing physical, psychological, and/or behavioral changes of sufficient severity to result in deterioration of interpersonal relationships and/or interference with normal activities" (Reid, 1985, p. 5). Consistent with criteria specified in the *Diagnostic and Statistical Manual of Mental Disorders* (American Psychiatric Association, 1994), this definition would exclude women with mild premenstrual symptoms (e.g., skin changes, edema, food cravings) that rarely disrupt normal functioning. It would, however, include more severe symptoms (e.g., irritability, depression, mood swings) if they seriously impair occupational and social activities (Schnurr, 1989).

Estimates, cited by Hardie, of the percentage of women who experience one or more symptoms at a level so severe and disabling as to be classified as suffering from PMS typically range up to 5 percent (e.g., Reid, 1991). Accepting this estimate, the prior odds that a randomly chosen ovulatory female does suffer from PMS is, thus, .052 (e.g., .05/.95), or 1 in 19 (Murphy, 1987). Even if Hardie's 283 ovulatory female employees qualified as a random sample, only some 15 (283/19=14.8) would likely qualify as PMS sufferers. Consequently, given the absence of specific inclusion and exclusion criteria in the Hardie study, sample-size adequacy for analytical purposes is an issue as error ranges may have been so large as to camouflage real effects due to the specific forces under consideration (Ferber, 1977).

Symptom Reporting

To the above consideration, it also should be noted that Hardie's reliance on a self-report retrospective instrument to gauge the impact of cyclic changes over a one-week period is likewise problematic. Prevalence rates based on retrospective data have been shown to be highly questionable, generally overestimating symptoms due to errors in recall. Symptoms occurring near menstruation are remembered, whereas those occurring during the intermenstrual phase are forgotten. Indeed, some 50 percent of women who report a history of premenstrual changes do not corroborate such reports when they complete daily diaries over multiple months (Rubinow & Roy-Byrne, 1984). For this reason, Rubinow and Roy-Byrne (1984) hold that "prospective longitudinal symptom reporting is the only acceptable way of demonstrating a relationship between mood changes and menstrual cycle phase" (p. 165).

Reid (1991) likewise states that prospective daily ratings are essential for confirming the severity and timing of symptoms so as to confirm a diagnosis. In this respect, Brooks-Gunn (1986) has stressed that the existence of premenstrual symptoms is an entirely different phenomenon from the designation of a premenstrual syndrome. By not addressing the question of symptom timing and failing to measure symptom severity, Hardie's findings fail to differentiate between symptom occurrence and symptom exacerbation (i.e., degree of impairment). This oversight is confounded by the fact that (a) PMS symptoms do not necessarily subside at the onset of menstruation and, thus, PMS sufferers may be symptomatic at other times of their menstrual cycle (Rubinow & Roy-Byrne, 1984) and (b) menstrual symptoms can vary throughout life, as well as from cycle to cycle (Brooks-Gunn, 1986). In recognizing these facts, a National Institute of Mental Health panel has accordingly recommended that a PMS diagnosis be made only when there is a 30 percent or more increase in the intensity of symptoms (e.g., depression, anxiety, irritability) in the seven days before menses as compared to the seven days afterwards (Hamilton, Parry, Alagna, Blumenthal, & Herz, 1984). Furthermore, it is recommended that the intermenstrual baseline be set over at least three ovulatory menstrual cycles to permit discrimination of random variability in symptom expression (Rubinow & Roy-Byrne, 1984). Assessment across cycles can also provide information on degree of symptom fluctuation and severity from one cycle to the next and, thus, the consistency and variability of the PMS state.

Risk Factors for Premenstrual Symptoms

In addition to Hardie's general failure to address the preceding concerns, a further unresolved methodological issue relates to risk factors that influence an individual's vulnerability to premenstrual symptomatology. Whereas the women included in Hardie's sample completed various self-report measures pertaining to health, work, and stress variables, no attempt was made to screen subjects for potential risk factors. For instance, the use of psychoactive medications, hormonal preparations (including oral contraceptives), mineral or vitamin supplements, and nonsteroidal antiinflammatory drugs are all considered possible PMS confounds. Moreover, women with irregular menstrual cycles (< 21 or > 35 days) are generally excluded from PMS samples (cf. Rivera-Towar & Frank, 1990). Furthermore, PMS symptoms have been shown to vary according to chronologic age, gynecologic age (i.e., sexual maturity), parity (i.e., having borne offspring), menstrual flow characteristics (i.e., intensity and duration), marital status, and various personality traits (see, e.g., Logue & Moos, 1986). A homogeneous, well-characterized sample is, thus, necessary to differentiate changes attributable to PMS from concomitants entrained to these changes. On this count, the meaningfulness of Hardie's results is, hence, suspect.

CONCLUSION

The preceding issues are obviously not unique to the Hardie study. Despite some 65 years of research (Richardson, 1995), numerous gaps exist in our understanding of PMS. PMS research is difficult not only due to the methodological issues noted, but because of a lack of agreement on the syndrome's definition and a failure to differentiate premenstrual symptoms from the more specific premenstrual syndromal condition. We have previously offered suggestions for future research into the effects of PMS on workplace behavior (Phillips & Bedeian, 1989) and will, thus, not do so again. Recognizing the difficulty of PMS research, we do applaud Hardie's effort to extend previous findings concerning the effects of PMS on job performance. We nevertheless are not yet convinced that the scientific evidence is sufficiently strong to conclude unequivocally that PMS does or does not affect workplace behavior. Like other areas of uncertainty and confusion, numerous unanswered questions remain. The lack of definitive answers results, in part, from researchers failing to avoid such methodological inadequacies as those cited. It is, thus, our hope that these comments will hereby inform future researchers contemplating work in this area.

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